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## **THE STATE OF CELLULAR IMMUNITY IN PATIENTS WITH KIDNEY DAMAGE AFTER THE SARS-COV2**

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**Resume.** Acute kidney damage in patients after SARS-CoV2 occurs against the background of a low number and activity of CD4+ cells and a high value of CD8+, CD19+ cells, which ultimately leads to impaired regeneration of individual parts of damaged nephrons, stimulating the growth and accumulation of circulating immune complexes and changing their antigenic structure. High values of phagocytic number and index may indicate the continued destruction of the basement membranes of nephrons, which ultimately leads to a decrease in the functional volume of the kidneys with the development of chronic renal failure.

**Keywords:** kidneys, cellular immunity, SARS-COV 2.

**Relevance.** To date, it has been established that the direct pathological effect of the SARS-CoV-2 coronavirus on the kidneys occurs through the receptors of the ACE2 organ, an angiotensin converting enzyme. Progressive application with the protein component of the receptor leads to the destruction of the renal parenchyma. Mediocre pathological effects occur through disorders in the immune system as a result of a so-called cytokine storm (1,3,5,7,31,32,33,34,35,36,37,38,39,40).

SARS-CoV-2 affects both the innate and acquired immune response of the host. SARS-CoV-2 can induce an immune response in two phases: an early specific acquired immune response to eradicate the virus and inhibit disease progression, and uncontrolled inflammation as a responsible mechanism for ARDS. The virus spreads and affects tissues with ineffective immune responses (2,4,6,8,10,41,42,43,44).

Necrosis or apoptosis of T cells promotes the release of a cytokine storm leading to a reduction of T cells (9,11,13,15), especially in cases with severe disease, lower circulating CD4+ and CD8+ T cells and higher, IL-10 and tumor necrosis factors- $\alpha$  (TNF $\alpha$ ) (12,14,16,18,20,22). Therefore, systemic inflammation harms viral clearance, contributing to the depletion of T cells (17,19,21,23,25,45).

Almost all patients with SARS-CoV-2 developed lymphopenia as an important marker of immune system disorders (24,26,28,30). Kidney macrophages play a key

role in immune defense, as they are the predominant cells interacting with virus targets and can activate phagocyte and chemokine signaling [27,29]. In addition, the cytopathic effect of the SARS-CoV-2 virus can directly damage renal tubular cells during infection and replication stages, spreading a complex immune response. In addition, the chemokine network, activation of complement cascades and coagulation play a potential role in the development of PP in patients with SARS-CoV-2.

**The purpose of the study.** To study the state of cellular immunity in patients with kidney damage after undergoing SARS-COV 2.

**Research materials and methods:** The paper presents information on the comprehensive examination and treatment of 62 patients with kidney damage who underwent SARS-CoV-2. The distribution of patients was carried out on the basis of a prospective targeted open randomized trial.

The period of research and collection of clinical material began in the second quarter of 2020 and ended in December 2023. At the same time, during the period from April to August 2020, the clinic also functioned as a specialized covid center, with the involvement of specialists from all directions in accordance with quarantine requirements. All patients were grouped into one main group.

The criteria for inclusion of patients in the main group were: the age of patients not younger than 20 and not older than 75 years; the presence of a history of coronavirus infection, with severe course, with signs of kidney damage during treatment; preservation of signs of kidney disease (proteinuria, albuminuria, micro- or macrohematuria, decreased glomerular filtration rate, high creatinine values and urea in the blood, etc.); the presence of a negative result of a PCR test for SARS-CoV-2 during hospitalization in our clinic; availability of the patient's voluntary informed consent to participate in a clinical trial.

In a comparative assessment of clinical and immunological changes, data from 20 healthy individuals recognized by the medical commission as absolutely healthy were used. All of them were combined into a control (reference) group.

The criteria for the inclusion of volunteers in the control group were: the age of patients from 20 to 50 years; the absence of any chronic somatic or mental diseases; the mandatory conclusion of the medical commission on a healthy state during the last 3 months before the study; the absence of a history of diseases from the urinary system; the absence of a history of SARS-CoV-2; the presence of a negative result of the PCR test for SARS-CoV-2; the presence of voluntary informed consent to participate in a clinical trial.

All patients in the main group were divided by stages of kidney damage after suffering SARS-CoV-2, which were also recommended by KDIGO.

Kidney damage was typical mainly for mature and elderly patients, while acute kidney damage after SARS-CoV-2 was typical for younger age.

Male patients prevailed (67.7%), and among both patients of the first and second subgroups.

Immunological studies were performed at the Bukhara branch of the Institute of Human Immunology and Genomics.

**Results and their discussion.** The study of the state of cellular immunity in patients with kidney damage after undergoing SARS-CoV2 was mainly aimed at assessing their activity against viruses. At the first stage, we studied the dynamics of changes in leukocytes in the blood. The general dynamics of leukocyte levels among patients with kidney damage after SARS-CoV2 was characterized by the presence of leukocytosis. Thus, the average value of leukocytes in the blood for the entire study period in patients with kidney damage after SARS-CoV2 was  $9.16 \pm 1.08 \times 10^9/l$  [CI:8.08; 10.08]. The maximum level of leukocytosis was noted among patients of the first subgroup before treatment [CI:10.47; 13.79], which was 2.9 times higher than the reference values ( $p < 0.05$ ). Even after the treatment in patients of the first subgroup, the level of leukocytosis was 2.75 times higher [CI:11.28; 12.22] than the level of the reference value [CI:4.08; 4.44]. In patients of the second subgroup, when contacting our clinic, the leukocyte level corresponded to a range of physiological values [CI:6.63; 7.67]. By the end of the treatment, this indicator decreased by 1.3 times compared to the period when patients were admitted to our clinic [CI:3.93; 7.25]. The number of neutrophils in patients with kidney damage after undergoing SARS-CoV2 was characterized by the presence of high values, although there was a relative decrease in the dynamics of the treatment. The average level of neutrophils in patients over the entire period of the study was  $68.89 \pm 1.91\%$  ( $p < 0.05$  relative to the reference values). At the same time, in patients before treatment, its level was equal to  $70.16 \pm 2.01\%$  ( $p < 0.05$  relative to the reference values), and after treatment they decreased to  $67.62 \pm 1.77\%$  ( $p < 0.05$  relative to the reference values).

The ranges of the confidence interval in the dynamics of the treatment ranged from [CI: 68.11; 72.22] before the treatment and within [CI: 65.86; 69.4] after the treatment. The average level of this indicator was [CI: 66.98; 70.81].

A separate analysis of the dynamics of changes in the number of neutrophils in the blood, depending on the time of admission to the clinic, showed that in patients of the first subgroup, the difference between the number of neutrophils before

treatment [CI: 67.81; 70.97] and after treatment [CI: 67.55; 72.23] was not significant, whereas in patients of the second subgroup, significant changes in according to this indicator, before [CI: 68.4; 73.46] and after [CI: 64.16; 66.56] treatment ( $p < 0.05$ ). The average number of neutrophils among patients of the first subgroup was  $69.64 \pm 1.96\%$ , and among patients of the second subgroup it was less -  $68.15 \pm 1.87\%$ .

The number of eosinophils over the entire period of the study in patients with kidney damage, after undergoing SARS-CoV2, averaged  $2.61 \pm 0.89\%$  [CI: 1.72; 3.51], exceeding the reference value by 1.2 times [CI: 1.64; 2.64] – Table 3.3. At the same time, the number of eosinophils in patients of the main group in The period of treatment at the clinic was characterized by an increase in this indicator of  $2.9 \pm 0.62\%$  [CI: 2.28; 3.52] compared with the reference values ( $2.14 \pm 0.5\%$ ;  $p < 0.05$ ).

In a separate analysis of changes in the level of eosinophils in the blood of patients of different subgroups, we noted relative eosinophilosis after a course of treatment among patients of the first subgroup [CI: 1.61; 0.33]. It should be noted that the initial value of blood eosinophils in patients of the first subgroup upon admission to our clinic was 1.3 times less than the reference [CI: 1.64; 2.64] values ( $p < 0.05$ ). After the treatment, in patients of the first subgroup, there was an increase in the number of eosinophils [CI: 2.58; 3.74], which exceeded the reference value by 1.5 times. At the same time, a 2-fold increase in eosinophils after treatment was also noted by us in relation to the period before the start of treatment [CI: 1.28; 1.94] in patients of the first subgroup ( $p < 0.05$ ). Thus, the average value of eosinophils among patients of the first subgroup for the entire period of the study was  $2.39 \pm 0.46\%$  [CI: 1.93; 2.84], which was lower than the overall average value [CI: 1.72; 3.5].

In patients of the second subgroup, the average value of eosinophils, which was  $2.84 \pm 0.47\%$  [CI: 2.37; 3.31], exceeded this indicator in patients of the first subgroup by 1.2 times [CI: 1.93; 2.84] and the index of reference values [CI: 1.64; 2.64] by 1.3 times.

When patients went to the clinic, before the start of treatment, a high value of this indicator was revealed [CI: 3.27; 5.09], which exceeded the reference values by an average of 1.9 times ( $p < 0.05$ ), the values of patients of the first subgroup before the start of treatment by 2.6 times ( $p < 0.05$ ) and the values of patients of the first the subgroups after treatment increased 1.3 times ( $p < 0.05$ ). We deliberately focus on this fact, since the level of eosinophils in the blood of patients of the second subgroup

before the start of treatment exceeded (by 1.4 times;  $p < 0.05$ ) the average value of this period for all patients with kidney damage after undergoing SARS-CoV2.

After the treatment, patients of the second subgroup have a mirror image of the reverse pattern in the change in the number of eosinophils in the peripheral blood. The number of eosinophils in patients of the second subgroup after treatment [CI: 1.47; 1.53] decreased by 2.8 times ( $p < 0.05$ ), compared with the previous study period. Compared with the same period of the study of patients of the first subgroup, there was also a 2.1-fold decrease in the number of eosinophils ( $p < 0.05$ ). The share of this indicator in the share of the total average value of patients in the main group after treatment was 64.4%, and among patients in the second group – 52.8%.

Thus, the increase in the number of eosinophils noted by us in chronic kidney damage after SARS-CoV2 is characterized by a decrease below the reference value after treatment, whereas in patients with acute kidney damage after SARS-CoV2, the reverse pattern occurs, characterized by an initially low number of these cells with gradual growth after treatment.

The average number of monocytes in peripheral blood in patients with kidney damage due to SARS-CoV2 was  $5.08 \pm 1.25\%$  [CI: 3.83; 6.32], which was 1.4 times higher than the reference values ( $3.73 \pm 0.51\%$  [CI: 3.22; 4.24]) ( $p < 0.05$ ).

monocytes in patients with kidney damage after undergoing SARS-CoV2 at the initial visit to our clinic equated to  $5.05 \pm 0.63\%$  [CI: 4.42; 5.68], and after treatment this indicator remained almost unchanged and amounted to  $5.10 \pm 1.86\%$  [CI: 3.24; 6.96].

In patients with acute kidney injury after SARS-CoV2, the average content of monocytes in peripheral blood was  $4.57 \pm 0.59\%$  [CI: 3.99; 5.16], which was 1.22 times higher than the reference values. Upon admission to the clinic, the number of monocytes in peripheral blood [CI: 4.03; 4.93] exceeded the reference value by 1.24 times. The number of monocytes in the peripheral blood of patients of the first subgroup upon admission to the clinic was 1.25 times lower than in patients of the second subgroup during this study period. The proportion of the average number of monocytes of patients before the start of treatment measures was 44.4%. The same level was noted in relation to the total average value of peripheral blood monocytes of the entire main group of patients.

After the treatment, the number of monocytes in peripheral blood increased slightly [CI: 3.94; 5.38] compared to the previous study period [CI: 4.03; 4.93], whereas among patients of the second subgroup there was an inverse pattern of changes (decrease in the number of monocytes from  $5.62 \pm 0.81\%$  to  $5.54 \pm 3.0\%$ ).

Thus, the overall dynamics of changes in the number of monocytes in peripheral blood was characterized by an unreliable increase after treatment. At the same time, a similar trend of changes was noted among patients with acute kidney injury after undergoing SARS-CoV2, which characterizes the pathological process as the determining mechanism of changes in the structure of leukocytes.

The pattern of lymphocyte changes in CD3<sup>+</sup> cells was almost identical in patients with various forms of kidney damage after undergoing SARS-CoV2. It was characterized by a decrease in the value in patients compared with the control data. Thus, the average value of the total number of CD3<sup>+</sup> T lymphocytes was  $61.87 \pm 3.0\%$  [CI: 58.88; 64.87], which was  $5.86 \pm 1.97\%$  less than the reference values [CI: 62.76; 72.7]. At the same time, the number of CD3<sup>+</sup> T lymphocytes in patients before treatment was lower ( $61.68 \pm 3.62\%$ ; [CI: 58.06; 65.3]) than in patients after treatment ( $62.07 \pm 2.37\%$ ; [CI: 59.7; 64,44]), which indicates a positive effect of methods for correcting kidney damage in patients who have undergone SARS-CoV2 in the form of CD3<sup>+</sup> cell expression.

Separate analysis, depending on the timing of treatment in our clinic of patients with kidney damage after SARS-CoV2, low average values of lymphocytes in patients with acute pathological process ( $60.92 \pm 3.39\%$ ; [CI: 57.53; 64.30]) can be noted than in patients with chronic pathological process ( $62.83 \pm 2.61\%$ ; [CI: 60,22; 65,43]).

In patients of the first subgroup, the number of CD3<sup>+</sup> cells in peripheral blood was higher before treatment [CI: 57.02; 65.32] than after replacement therapy [CI: 58.04; 63.28]. In both cases, these indicators were lower than the reference values by  $6.56 \pm 1.32\%$  and  $7.07 \pm 2.85\%$ , respectively. At the same time, in patients of the second subgroup, the tendency to change the number of CD3<sup>+</sup> cells corresponded to the general average, was higher than in patients with acute kidney injury after SARS-CoV2 and was characterized by an increase in CD3<sup>+</sup> cells ([CI: 59.09; 65.27] before treatment and [CI: 61.35; 65.59] after treatment).

We obtained a relatively similar dynamics of changes when studying the number of subpopulations of T helper lymphocytes by CD4<sup>+</sup> cells.

The average level of CD4<sup>+</sup> T helper cells among patients with kidney damage after SARS-CoV2 was lower ( $34.58 \pm 1.34\%$ ) than the reference values ( $35.26 \pm 1.32\%$ ), although the confidence interval range did not have such a high difference ([CI: 33.94; 36.58] in the control group and [CI: 33.24; 35.92] the average level of the main group of patients). At the same time, between patients with kidney damage after SARS, the average value was before ( $34.41 \pm 1.29\%$ ; [CI: 33.13; 35.7]) and after

( $34.75 \pm 1.4\%$ ; [CI: 33.35; 36,15]) treatment did not have a significant difference, which was due to different phases of the pathological process ( $p > 0.05$ ).

The average value of CD4+ T-helper cells among patients with acute kidney injury after undergoing SARS-CoV2 was  $35.43 \pm 1.51\%$  [CI: 33.92; 36.94], whereas among patients with Over a longer period of the pathological process, this indicator was lower and amounted to  $33.73 \pm 1.18\%$  [CI: 32.56; 34.91].

When conducting a separate analysis, it can be noted that in acute kidney injury, in patients who underwent SARS-CoV2, the number of CD4+ T helper cells increased [CI: 34.74; 37.48], which was the maximum value compared with the general main group [CI: 33.13; 35.7]. A decrease in CD4+ cell T-helper cells was characteristic of patients with kidney damage after undergoing SARS-CoV2 [CI: 31.51; 33.91], which acquired a chronic course. Renal replacement therapy in patients of the first subgroup led to a decrease in the number of CD4+ T helper cells in peripheral blood [CI: 33.1; 36.4], and hemodialysis in the second subgroup of patients ended with an increase in the number of CD4+ T helper cells [CI: 33.6; 35.9]. In other words, the acute inflammatory process occurred against the background of suppression of T-helpers, whereas the chronic one was stimulated. However, in both cases, the data obtained indicated that the reference values were not reached, which was typical for kidney damage after SARS-CoV2. After the treatment, the level of CD4+ cells in both subgroups becomes the same.

The average number of cytotoxic CD8+ T lymphocytes in patients with kidney damage after SARS-CoV2 was almost at the level of reference values, which was determined by the similarity of fluctuations in the confidence interval ([CI: 19.84; 21.54] reference values and [CI: 19.02; 21.22] in the main group of patients).

The average number of CD8+ cells in the control group was  $20.69 \pm 0.85\%$ , whereas in patients of the main group it was  $20.12 \pm 1.1\%$ .

In patients of the main group, the number of CD8+ cells when contacting our clinic was higher than  $20.37 \pm 1.04\%$  [CI: 19.33; 21.41] than in subsequent periods of therapy  $19.87 \pm 1.17\%$  [CI: 18.71; 21.04], that is, against the background of replacement therapy, the number of cytotoxic T lymphocytes CD8+ cells it decreased, reaching a level below the reference values.

We noted higher values of CD8+ T cells among patients with acute kidney injury after undergoing SARS-CoV2 [CI: 19.93; 21.55]. This value was higher than the reference value [CI: 19.84; 21.54] and the values of patients in the second subgroup [CI: 18.73; 21.27]. After the therapy, the number of CD8+ T cells in patients of the

first subgroup decreased [CI: 18.08; 20.0], whereas in patients of the second subgroup it increased [CI: 19.33; 23.07].

The average number of CD19+ B lymphocytes in patients with kidney damage after SARS-CoV2 was  $10.83 \pm 1.12\%$  [CI: 9.72; 11.95], which was higher than the reference values ( $9.0 \pm 0.79\%$  [CI: 8.21; 9.79]). The variance of B lymphocyte values among patients with kidney damage was low at the time of admission to the clinic ( $10.53 \pm 1.0\%$  [CI: 9.54; 11.53]), followed by an increase to  $11.13 \pm 1.24\%$  [CI: 8.21; 9.79] as a result of substitution therapy. In general, patients with kidney damage after SARS-CoV2 were characterized by an increased value of CD19+ cells. In patients with acute kidney injury after undergoing SARS-CoV2, the level of CD19+ cell elevation was higher than among patients with a chronic pathological process. The number of B lymphocytes increased after the treatment.

The phagocytic number in patients with kidney damage after undergoing SARS-CoV2, both when contacting the clinic and during treatment, was 2.35 times higher than the reference values ( $p < 0.05$ ) and 2.13 times ( $p < 0.05$ ), respectively.

In patients of the second subgroup, this difference was more pronounced upon admission to the clinic (3.18 times;  $p < 0.05$ ) and tended to decrease after therapy. In patients of the first subgroup, changes in the phagocytic number were of a mirror-like nature, that is, after treatment, they increased.

The phagocytic index in patients of the main group, which was  $54.18 \pm 3.09$  CU [CI: 51.09; 57.26], was higher than the reference values.

At the same time, in the dispersion value, the reserve capabilities of phagocytes were relatively high in patients before replacement therapy and amounted to  $54.6 \pm 2.93$  CU [CI: 51.67; 57.53]. After treatment, the phagocytic index decreased to  $53.76 \pm 3.24$  CU [CI: 50.52; 57.0].

On the day of hospitalization of patients to the clinic, relatively high values of the phagocytic index were detected by us in patients of the first subgroup. The same value was noted by us among patients of the second subgroup. After treatment in patients of both the first and second subgroups, the number of phagocytic index decreased in an unreliable value.

The average number of phagocytic index in patients of the first subgroup was  $54.3 \pm 3.99$  cu [CI: 50.31; 58.28], and in patients of the second subgroup -  $54.06 \pm 2.19$  CU [CI: 51.88; 56.25].

The reference values of circulating immune complexes were  $33.47 \pm 1.4$  cu [CI: 32.07; 34.87], whereas in patients of the main group they increased to  $57.25 \pm 4.96$  cu [CI: 52.28; 62.21].

The prevalence of circulating immune complexes was noted among patients before treatment -  $62.52 \pm 5.37$  CU [CI: 57.15; 67.89]. After replacement therapy, the level of circulating immune complexes in the blood decreased to  $51.98 \pm 4.56$  CU [CI: 47.42; 56.53], although it remained 1.55 times higher than the reference values ( $p < 0.05$ ).

The highest values were found among patients with acute kidney injury after SARS-CoV2 up to  $64.94 \pm 5.7$  CU [CI: 59.25; 70.64], which was 1.94 times higher than the reference values ( $p < 0.05$ ). As a result of the treatment, the decrease in the concentration of circulating immune complexes was insignificant – by  $9.48 \pm 1.17$  cu. We deliberately focus on this fact, since in patients of the second subgroup, the relatively low values of circulating immune complexes in the blood at admission to the clinic, after treatment, decreased by  $11.60 \pm 2.46$  cu.

### **CONCLUSION.**

1. Acute kidney damage in patients after SARS-CoV2 occurs against the background of a low number and activity of CD4+ cells and a high value of CD8+, CD19+ cells, which ultimately leads to impaired regeneration of individual parts of damaged nephrons, stimulating the growth and accumulation of circulating immune complexes and changing their antigenic structure.
2. High values of phagocytic number and index may indicate the continued destruction of the basement membranes of nephrons, which ultimately leads to a decrease in the functional volume of the kidneys with the development of chronic renal failure.

### **LITERATURE**

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